viable kidney tissue remaining is situated at both poles, and in the small compressed region of the hilus. The solid mass compressing the kidney appears to be of a pus-like nature in many areas.

Microscopically, this is a large kidney abscess which has been walled off by a coarse layer of chronic granulation tissue heavily overrun by numerous polymorphonuclears, plasma cells, lymphocytes and a few eosinophiles. Within the abscess there are large numbers of inflammatory cells and exudate. The abscess is markedly compressing kidney structure, and the adjacent tissues show a heavy inflammatory cell infiltration. Many of the tubules are of a necrotic nature, while the remainder are showing degenerative changes. A few are dilated and contain pus. Towards the cortex the glomeruli are congested and a number have undergone complete fibrosis. The interstitial tissue throughout is also infiltrated, both diffusely and focally, with large numbers of lymphocytes, plasma cells and polymorphonuclears intermingled. There is no evidence of malignancy nor of specificity of the inflammatory process.

SUMMARY

1. A proved case of kidney carbuncle is presented.

2. The causative organism was *Staph. aureus*.

3. No primary focus was detected.

4. Diagnosis was made after operative exposure.

5. Patient recovered after primary nephrectomy.

I am indebted to Drs. James Bell and W. L. Robinson for their excellent pathological reports.

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A SULCUS TUMOUR

BY LOUIS J. BRESLIN

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W.G., a white male, 66 years of age, first consulted me in 1934 because of a mild diabetic condition.

Past medical history.—Pneumonia in 1923; suprapubic prostatectomy for benign hypertrophy of the gland in 1930.

Family history.—Not significant.

Physical examination at that time revealed a healthy looking man, 5 ft. 11 inches in height and weighing 174 lbs. The fundi exhibited slight pallor and some tortuosity of arteries with grade 1 narrowing of their lumen. The veins were nowhere compressed. Vision, hearing, etc., excellent. The tonsils were enlarged and looked diseased. The mouth was edentulous, 2 plates being worn, and a large lipoma was seen at the back of the neck. The heart showed evidence of some hypertrophy and dilatation, the apex being outside the left nipple line; short systolic murmur could be heard here; an occasional extrasystole was present. Blood pressure in both arms 160/90 mm. of Hg.; the peripheral vessels were definitely sclerosed. Fluoroscopy of the chest revealed a shadow at the left apex extending to the first rib on that side; the percussion note was but little impaired here; breath sounds normal. Physical examination was otherwise negative. The urine had specific gravity of 1.024, showed albumin 1 plus, sugar 2 plus, white blood cells 3 plus, and occasional coarse granular casts. Fasting blood sugar 0.167; non-protein nitrogen 38, cholesterol 220, van den Bergh 0.4 units. Red blood count, 4.2 million, white blood count 8,000, Hgb. 82 per cent (Sahli), smear normal.

The patient pursued a satisfactory course until January, 1936, when he exhibited evidences clinically and electrocardiographically of posterior coronary occlusion. Satisfactory convalescence ensued at the end of two months.

In October, 1937, he began to experience severe pain in the left scapular region, spreading to the shoulder and later on the arm. This became aggravated in paroxysms, especially whilst moving the affected limb. The pain gradually became more persistent, extended along the inner aspect of the arm, finally reaching the outer 2 or 3 digits. No atrophy, sensory or colour changes were manifest. X-ray films revealed the shadow at the left apex which I had previously noted in 1934. Moderate osteoarthritic changes were evident throughout most of the spine. The patient went south in January of 1938. His pain became progressively worse so that he lost weight and strength. He developed about this time a left-sided Horner's syndrome, and when seen by Dr. E. S. Nichol, of Miami, was considered to be a case of superior pulmonary sulcus tumour.

Upon his return to Toronto in March of 1938 he presented an emaciated appearance, and was manifestly suffering excruciating pain. The findings were complete Horner's syndrome; the skin of the face, upper arm and adjacent chest wall was dry and flushed, whereas the skin below nipple line perspired profusely. Left arm showed mild degree of weakness in all muscle groups; slight atrophy was detectable in the muscles of the forearm as well as those of the hand. Reflexes present and equal in both upper extremities. No definite sensory changes were detected. Lumbar puncture yielded clear fluid, pressure of 110 mm. water, free rise and fall on Queckenstedt. Kahn test negative; total protein 47 mg. per 11 c.c.; colloidol gold unchanged; 2 lymphocytes per c.mm.

Re-check x-ray films of chest, cervical and thoracic vertebræ, ribs, etc., were taken (see Fig. 1). Several radiologists who were consulted referred to "ill defined area of increased density in the left upper chest, lower border of lesion roughly paralleling lower border of first rib. This area, of fairly homogeneous density. No evidence of destruction of bone in any of the ribs, cervical or thoracic spine. No displacement of trachea." One radiologist considered the sternal end of the 1st left rib to be involved.

The patient went down hill rapidly and requested exploratory operation and if possible rhizotomy. The former procedure was performed at St. Michael's Hospital, Toronto, on May 1, 1938. A hard apical pulmonary tumour underlying and attached to the pleura was exposed. The mass pressed upon and distorted the brachial plexus. Biopsy material obtained revealed an anaplastic form of carcinoma (see Fig. 2). Pneumonia and cardiac failure set in. Exitus occurred on the fourth day after operation. Permission for autopsy was refused.

This case is reported for two reasons. (1) To draw attention again to a syndrome first described by Pancoast in 1924, *viz.*, radiographic evidence of dense shadow at the extreme apex of one lung; erosion of one or more upper ribs, usually close to their vertebral attachment; pain at first paroxysmal, later continuous, commonly commencing at the outer border of the scapula, corresponding shoulder and upper chest regions, oft-times extending into the axilla, inner side of arm, but rarely beyond the elbow; weakness; atrophy of the smaller muscles of hand; development of Horner's syndrome. It is interesting

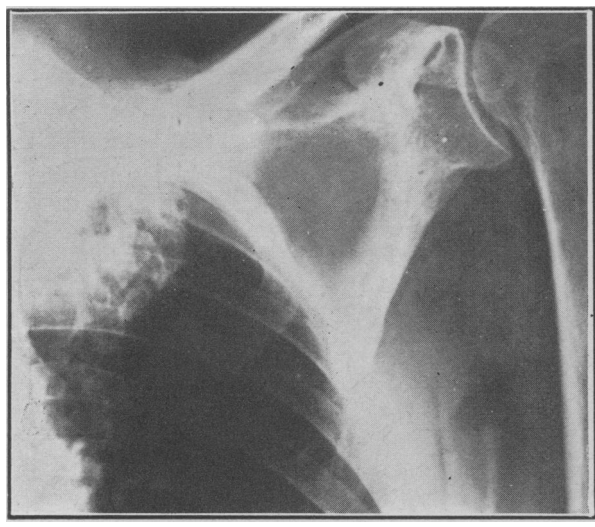

Fig. 1

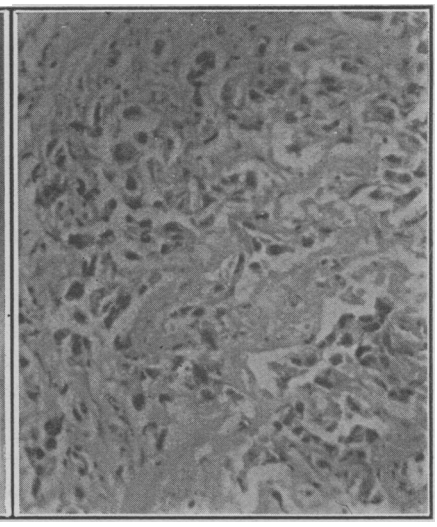

Fig. 2

to note that Hare[5] described a similar case in which he found at autopsy a hard nodular mass extending up to the origin of the brachial plexus. (2) Because of the fact that this apical shadow was noticed in September of 1934, being interpreted at that time as a pleural thickening. It is unfortunate that no photographic records were taken then. So far as I could judge, there was no noticeable increase in size of alteration in nature of this shadow. Feldman, Davidsohn and Danelius[2] suggest that dense pleural apical adhesions may precede development and growth of such a tumour and facilitate its expansion towards the thoracic wall and spinal column.

Pancoast was of the opinion that sulcus tumours originated from the epithelium, of one of the lower branchial clefts, but not from the lungs, pleura, ribs, or mediastinum. It is now believed that the entire symptomatology can be produced by any inflammatory or tumour mass, either primary or secondary arising in this location. Although apical bronchogenic carcinoma can therefore produce this clinical picture it is relatively unusual for it to do so. When such is the case there is striking absence of the usual symptoms of lung neoplasm, *viz.*, cough, mucoid expectoration, hæmoptysis, etc. It has, further, become increasingly apparent that invasion of rib and vertebræ is not as constant a finding as Pancoast considered it to be.

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Clinical and Laboratory Notes

STUCK SYRINGES

BY FREDERICK WERTHENBACH

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In every doctor's office there is the occasion when a syringe becomes stuck. Many methods and devices are in use to separate the syringe parts, some of which are costly, both in equipment and breakage. Most of the surgical firms have a metal syringe which fits the end of the syringe which has become stuck, and warm water may be forced into the stuck syringe. However, if the least pressure out of a straight line is put on this equipment the end of the syringe is usually broken. To overcome this breakage, and because of the original cost of the syringe provided by the surgical firms, I was of the opinion something very simple should be used, and I had this made for a few cents. It is as follows. An ordinary 20 gauge needle to fit our syringes was soldered into an old trocar tube, the end of which had threads which fit on an Imperial dental syringe. The equipment is about three inches long, is flexible, and the total cost was only a few cents. Any ordinary syringe may be used, the smaller the barrel the better, and two needles soldered together. This little invention is made necessary because surgical houses have overlooked the demand for a "like to like" adapter, *i.e.*, Luer to Luer, etc.